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Polyneuritis Cerebralis Ménièriformis

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POLYNEURITIS CEREBRALIS MÉNIÈRIFORMIS

Landry [1], in 1859, presented 10 cases which were characterized by paralysis beginning in the lower extremities and rapidly ascending to the trunk, upper extremity, neck, and head. Though sensory disturbances were in the majority of cases minimal, the author noted that sensibility and mobility could be equally involved. Two of the cases terminated fatally. Analysis of the 10 cases reveals that three forms of the process were described: (1) commencing in the lower extremity, 'becoming generalized without systematized sequence', and producing both motor and sensory changes; (2) commencing in the lower extremity, 'becoming generalized with a systematized sequence' and involving only motility; and (3) similar to (2), but including both motor and sensory involvement. In time the term 'Landry's paralysis' has been used by clinicians to describe only the first form.

Guillain, *Barré*, and *Strohl* [2] were the first observors to report spinal fluid changes in the disorder. In a description of 2 cases, published in 1916, they noted an elevated protein and *normal* cell count. The course of both cases was benign. In 1936 *Guillain* [3] described 10 further non-fatal cases with albumino-cytologic dissociation. He concluded that the criteria which differentiated the Guillain-Barré Syndrome from that of Landry were the involvement of both motility and sensibility, and benign prognosis of the former.

Guillain and *Barré* [4] modified their position in 1938 in conceding that under certain conditions (involvement of bulbar nerves) the process described by them could terminate fatally. An examination of Landry's [1] original paper reveals, further, that disturbances of sensibility were not uncommon in the cases which he described.

Haymaker and *Kernohan* [5] presented 50 cases of Polyneuritis and reviewed the literature. They concluded that the disease entities described by Landry and Guillain, Barré, and Strohl were different manifestations of the same process, and suggested use of the term 'Landry-Guillain-Barré Syndrome'. The authors further observed, that the spinal fluid changes, which had been considered necessary for the diagnosis of the Guillain-Barré Syndrome were not consistently present in the cases reviewed by them. Spinal fluid protein, though often elevated, was at times normal, and even reached below normal limits. The level tended to vary, depending upon the stage of the disease during which the determination was made, being often low initially, and becoming elevated in the later stages. The cell counts reported were also variable. Though normal to low cells were the rule, Pleocytosis was a not uncommon finding.

An examination of the literature reveals that three theories have been used to explain the mode of spread of Polyneuritis. Some authors [6–8] feel that the primary lesion is a vasomotor paralysis. Others [9–10] consider the Neuraxis the ascending

and descending pathway of spread. A third group [11–13] fells that the process is disseminated via the spinal fluid. In the absence of experimental evidence, the theories remain hypothetical.

In 1865 *Pellegrino-Lévi* [14] reported a case of 'Landry's Paralysis' which commenced in the cranial nerves and descended symmetrically to involve the nerves innervating the limbs and trunk. Since that time, the process, though most commonly confined to the Facial, has been described involving all cranial nerves.

Frankl-Hochwart [15] in 1895 described a case of cerebral Polyneuritis, whose symptomatology closely resembled that of Ménière's disease. The patient was a 40 year old male, who experienced an episode of headache and vomiting of sudden onset. Within a few hours he began to complain of tinnitus on the left side and vertigo, followed by diminished auditory acuity also on the left. The Romberg was positive, with a tendency to fall to the left. Three days later Facial paresis of the peripheral type was noted. Return to normal function was observed, excepting auditory acuity.

In 1898 *Hammerschlag* [16] presented a case of a 32 year old male, whose first complaints were of nausea, vertigo, and headache. The following nerves were involved in order of appearance of the lesion: Facial paresis on the right side, all branches of the sensory division of the right Trigeminal, and the right Acoustic. Because of exposure of the patient to cold preceeding the illness, the author assumed a 'rheumatic' aetiology. The appearance of an herpetic lesion during the course of the illness was also noted. The illness remitted with eventual exacerbation of the auditory symptoms.

Hammerschlag also reported upon a case previously described by *Kaufmann* [17]. The patient, a 34 year old male, first noted malaise, fever, and hedache of sudden onset. Five days later he complained of pain on the left side of the face and vertigo. Herpes Zoster blisters were noted in the area of the Maxillary division of the left Trigeminal nerve. Four days following this, paralysis of the left Facial nerve and diminished auditory acuity, also on the left, developed. Spontaneous healing occured.

Muck [18] described a similar case, which begann with vertigo, tinnitus, and pain on the left side of the face. Tests of auditory function revealed diminished acuity on the left side. The examinor was unable to elicit a response by means of caloric stimulation of the left Vestibular nerve. Three days following the appearance of the initial symptoms, paralysis of the left Facial nerve was described. Herpes Zoster was noted during the course of the illness.

Thornval [19] described three cases, two of which were he considered of 'rheumatic' aetiology, and the third of luetic. Two days following an episode of Angina, paralysis of the right Facial nerve was noted in a 43 year old female. One day later vertigo, tinnitus, and diminished auditory acuity on the right side appeared. Caloric stimulation of the Vestibular nerve was negative. Ten days later Herpetic blisters in the areas of the first two divisions of the right Trigeminal nerve were noted, though no pain was reported. Healing was spontaneous.

The second case involved a 42 year old female, whose illness began with pain in the left ear. Three days following this she complained of vertigo. Within 24 hours paresis of the left Facial nerve was noted, which was followed by tinnitus and diminished auditory acuity on the left side. Auditory and Vestibular tests substantiated the diagnosis.

In the above 2 cases vestibular function was more completely involved than auditory. Remission of auditory function, however, was more delayed in both instances.

Thornwal's third case was that of a 20 year old male, who was being treated with Salvarsan for a luetic infection acquired three months previously. Vertigo and tinnitus developed on the right side, and were followed by decreased auditory acuity and paresis of the right Facial nerve. Examination of the spinal fluid revealed a $\pm$ Wassermann, and protein determinations of 10 and 15 mgs%. Further anti-syphilitic therapy resulted in clinical cure, though caloric tests of vestibular function remained negative.

Zaviska [20] reported three cases of 'Polyneuritis cerebralis Ménièriformis', all of which began with unilateral Facial nerve paralysis, and were followed by auditory and vestibular disturbances.

Fischer [21] described 2 cases, the first of which involved a 30 year old female, who complained of pain in the left ear, vertigo, tinnitus, and headache. Auditory and vestibular studies confirmed involvement of both divisions of the eighth nerve on the left side. Subsequently, Herpetic blisters appeared in the area of the Maxillary division of the fifth nerve. Within a few days the right Statoacoustic and trigeminal nerves were similarly affected. At no time did Facial paresis occur. Healing was spontaneous, being first evident on the left side.

The second case was that of a 27 year old male, who first complained of tinnitus on the left side, which was shortly followed by similar symptoms on the right. Pain in the areas of distribution of both Ophthalmic divisions of the fifth nerve, and vertigo appeared. On the sixth day Herpetic lesions of the Lesser Occipital nerves were noted. Healing was complete within 3 weeks.

Veraguth [22] described a patient with Facial paresis, tinnitus, and auditory hypoacusis on the right side, and vertigo. Retrobulbar neuritis on the right later developed. He attributed the lesions to a luetic infection, and treated the patient with Salvarsan. One year following institution of therapy, he noted only a partial return of function of the Acoustic and Facial nerves.

Pekelsky [23] described a case which began with headache, fever, vertigo, vomiting, ptosis, and auditory hypoacusis on the left side. Subsequently, lesions of the left Olfactory, Trigeminal, Abducens, and Facial nerves developed. Following a short remission of symptoms, an exacerbation occured accompanied by weakness of the left arm and leg, diminished deep tendon reflexes in both limbs, and myoclonus of the left foot. Disturbances of sensibility on the left side, particularly of the face and arm were also reported. Healing was spontaneous.

Krivy [24] reported a case of a woman, who developed lesions of the Abducens, Facial, Statoacoustic, Glossopharyngeal, Vagus, and Spinal Accessory nerves, all on the right side, and the Oculomotor nerve on the left side subsequent to a miscarriage. He assumed an 'infectious-toxic' aetiology.

Analysis of the cases reviewed (Table 1) reveals that two-thirds of the patients were male. The average age was 32 (20–43) years. Prodomal symptoms included headache, nausea, vomiting, malaise, fever and facial and aural pain. In seven instances the eighth nerve was the first involved, in five it was the seventh, in one case both seventh and eighth simultaneously, in one, fifth and eighth simultaneously, and in one no

Table 1: *Review of Cases in the Literature*

Author	*Case*	*Age, Sexe*	*First Symptoms*	*First Nerve Involved*	*Subsequent Nerves Involved*	*Aetiology*	*Course*
Frankl-Hochwart	(1)	40, male	headache, vomiting	VIII L	VII L	?	Return to normal function except Acoustic
Hammerschlag	(2)	32, male	nausea, vertigo, headache	VII R	V R VIII R	Herpes Zoster	Remission with exacerbation of Acoustic
Kaufmann	(3)	34, male	malaise, fever, headache	V L Vestibular L	VII L Auditory L	?	Return to normal function
Muck	(4)	24, male	vertigo, facial pain, tinnitus	VIII L	VII L	Herpes Zoster	Return to normal function Facial last to heal
Thornval	(5)	43, male	Facial nerve paralysis	VII R	VIII R V 1–2 R	Herpes Zoster	Return to normal function Auditory last to heal
	(6)	42, female	aural pain	Vestibular L	VII L Auditory L	?	Return to normal function Auditory last to heal
	(7)	20, male	vertigo, tinnitus	VIII R	VII R	Lues (Herxheimer reaction)	Return to normal except Vestibular tests
Zaviska	(8)			VII	VIII	?	
	(9)			VII	VIII	?	
	(10)			VII	VIII	?	
Fischer	(11)	30, female	aural pain, vertigo, tinnitus	VIII L	V L VIII R V R	Herpes Zoster	Healing first on left side

	(12)	27, male	pain, tinnitus	Auditory L	Auditory R Vestib. R and L V, R and L	Herpes Zoster	Healing within 3 weeks
Veraguth	(13)	–	Facial paresis, tinnitus, audit. hypoacusis, vertigo	VII R VIII R	Optic R	Lues	Partial return of normal function of Auditory and Facial
Pekelsky	(14)	–	Fever, vertigo, ptosis, audit. hypoacusis	VIII L	I, VI, V, VII L L arm, leg, and trunk	?	Return to normal function
Krivy	(15)				VI, VII, VIII, IX, X, XI R, III L	?	

information was available. Seven cases presented lesions of the seventh and eighth nerves, three of the fifth, seventh, and eighth, two of the fifth and eighth, one of the second, seventh and eighth, one of the first, fifth, sixth, seventh, eighth, and entire left side of the body, and one of the third, sixth, seventh, eighth, ninth, tenth, and eleventh nerves.

In all instances in which the Facial and Statoacoustic, or the Facial, Statoacoustic, and Trigeminal were involved, the lesion was homolateral. In the two cases in which the Statoacoustic and Trigeminal were involved, with no Facial paresis, the lesion was bilateral. Herpes Zoster was implicated in five instances, Lues in two, and eight were of undetermined aetiology. In all cases recorded there was at least partial return of function. In five instances the Acoustic, in two the Facial, and in one the Vestibular nerve showed the least favorable prognosis. In no case did the disease terminate fatally.

In case 7 cerebrospinal fluid protein determinations of 10 and 15 mgs% were recorded. There was no data available on Leucocyte counts, either in the cerebrospinal fluid or peripheral blood, nor were there blood pressure values recorded.

Case report

The author will at this time present a case of similar symptomatology, which was seen in the Neurological Institute during the first half of 1962.

MB was a 66 year old white female, who was first seen in the out-patient department of the Neurological Institute in January 1962. She complained, at that time, of paresis of the mimic musculature and excessive lacrimation on the right side, of five days duration. She also complained of slight pain of the Occiput, radiating to her right ear, of fourteen days duration. The patient reported a similar episode on the left side two weeks prior to her first visit, the effects of which disappeared within a week. Four weeks preceding this she had a episode of Influenza.

The family history was non-contributory. The patient's past history included Brachialgia of many years duration, due to a Cervial Syndrome, and responsible to physiotherapy, Iridocyclitis, and Rosacea.

The general physical examination, exepting a blood pressure of 180/195 was not contributory. Neurological examination of the cranial nerves revealed consensual reaction of the isocoric pupils to light and accomodation, and normal ocular mobility. The patient was found to have a bilateral ageusia of the proximal two-thirds of the tongue. Paresis of all branches of the right Facial nerve was found. Examination of the left seventh, as well as the eighth, ninth, tenth, eleventh, and twelfth nerves revealed no neurological deficit. Muscle tone, mobility, and deep tendon reflexes of the extremities were normal, as were the sensory modalities of touch, pain, temperature, two-point discrimination, and vibratory-sense. The Babinski and Romberg were negative, and the gait normal. Tenderness of the Cervical vertebrae was noted.

Hematological examination revealed: Hb 86%, RBC 4.1, color index 1.04. WBC 4,200, with 36% Neutrophils, 3.5% Eosinophils, 1% Basophils, 50.5% Lymphocytes, and 9% Monocytes. The ESR was 7/17 and the Wassermann reaction nega-

tive. Electromyographic studies revealed fibrillation potentials and greatly diminished number of motor unit action potentials of the Orbicularis Oris and Frontalis on the right side. An Otorhinolaryngological consultant noted a normal Audiogram and normal reaction of the Vestibular nerves to thermal stimuli.

The patient was placed on Hydergin, Tofranil and Dianavit, and followed in the out-patient department.

During the following month her condition remained unchanged. In February 1962, she was admitted to the Neurological Institute as an in-patient. At that time she complained of anorexia and vertigo, and was found to have a positive Romberg, and an unsteady gait. Her blood pressure was 130/180. She remained normo to hypotensive during the entire admission. Otorhinological examination revealed diminished reaction of the right Labyrinth to thermal stimuli. The Audiogram was normal. Examination of the spinal fluid showed (Table 2): pressure 65 mm H_2O, 86 cells, 73 mgs% protein, Nonne +, Pandy ++, 72 mgs% sugar. Hematological studies again revealed relative lymphocytosis.

Table 2: *Cerebrospinal Fluid*

Date	*Cells*	*Protein*	*Sugar*
2/26	86	73 mgs%	72 mgs%
4/2	78	77 mgs%	
5/3	11	104 mgs%	

Two weeks following admission the patient began to complain of rapidly diminishing hearing. The Otological examination was repeated and showed bilateral auditory anacusis. The examiner was unable to stimulate either Labyrinth thermally. Deep tendon reflexes were diminished in all extremities. Examination of sensation was not possible because of deafness. Conduction studies of the peripheral nerves and electromyographic examination (Tables 3 and 4), indicated increasing involvement of the peripheral nervous system, being more pronounced initially in the upper extremity. Repeated spinal taps revealed moderate Pleocytosis and high protein values.

The diagnosis of infectious Polyneuritis was considered most likely, and the patient placed ob Prednison, Penicillin, and Streptomycin. Bacteriological and serological tests (Tables 5 and 6) were negative, excepting a positive Herpes complement fixation test. The possibility of Sarcoidosis was entertained. Supraclavicular node biopsy, however, was negative.

The patient became progressively somnolent and cachectic. In mid-April she was febrile and incontinent, and showed tachycardia and Cheyne-Stokes breathing. Deglutitive disturbances appeared. It was, at that time, thought that the ninth and tenth nerves were involved, but due to the patients somnolent state, this could not be objectified.

Table 3: *Electromyographic Studies*

Date	*Muscle*	*Fibrillation*	*Fasc. Pot.*	*MUAP*
1/31	Orbic. oris R	+	+	greatly decreased in number
	Frontalis R	(+)	+	greatly decreased in number
3/26	Orbic. oris R	++	–	? patient unable to cooperate
	Quadriceps R	–	–	
	Tibialis ant. R	–	–	
	Extensor dig. brev. R	–	–	
5/15	Orbic. oris R	++	–	? patient unable to cooperate
	Orbic. oris I	++	–	
	Tibialis ant. R	++	–	
	Quadriceps R	++	–	
	Gastrocnemius L	(+)	–	
	Inteross. dors. I L	(+)	–	
	Quadriceps L	+	–	

Table 4: *Nerve Conduction Studies*

Date	*Nerve*	*Muscle*	*Conduction velocity*	*Distal. Delay*	*Ampl.*
			m/sec	ms	mV
3/7	Median R	Thenar	40.6	4.8	9.2
	Ulnar R	Hypothenar	42.7	2.7	7.0
	Peroneal R	Ext. dig. brev.	35.0	5.0	5.7
5/15	Median R	Thenar	46.5	4.5	4.2
	Ulnar R	Hypothenar (elbow)	40.5	2.6	3.8
	Ulnar R	Hypothenar (upper arm)	55.5	–	2.8
	Peroneal R	Ext. dig. brev.	41.0	9.5	1.8

Table 5: *Bacteriological Tests*

Date	*Medium*	*Organism*	*Result*
2/27	stool	Poliomyelitis	negative
2/27	stool	Coxsakie	negative
2/28	blood	Listeria	negative
3/6	stool	Poliomyelitis	negative
3/6	stool	Coxsakie	negative

In May 1962 the patient showed flaccid paralysis of her entire right side. Electroencephalographic studies indicated a lesion in the left Temporal lobe. Carotisangiography revealed delayed filling of the branches of the left Internal Carotid artery. The changes were attributed to cerebral thrombosis.

The patient's condition continued to deteriorate until she died in August 1962 of respiratory insufficiency.

Obduction performed at the Institut of Pathology University of Basle revealed demyelination and inflammatory changes in the right Facial, both Statoacoustic, the Ulnar, the Median, and the Fibular nerves. Some Myelitis was also present. The

Table 6: *Serological Tests*

Date	*Medium*	*Organism*	*Test*	*Result*
1/26	serum	Trepon. pallid.	Wassermann	negative
			Citochol	negative
			Kahn	negative
2/27	serum	Leptospira	Agglutination	negative
2/27	serum	Toxoplasma	Dye test	negative (1:64)
2/27	serum		Cold agglut.	negative
2/27	serum	Typhoid	Agglutination	negative
		Paratyphoid B		negative
		Gärtner Enteritis		negative
		Bac. Dysent.		negative
		Brucellosis		negative
2/27	serum	Q-Fever	Compl. fix.	negative
		Influenza A		negative
		Influenza B		negative
		Ornithosis		negative
		Adeno-Virus		negative
		Lymphocity Choriomening.		negative
		Herpes		1:32
2/27	serum	Listeria	Agglutination	negative
		Listeria	Compl. fix.	negative
2/27	serum	Poliomyelitis, type I	Compl. fix.	1:24
		type II		1:24
		type III		1:24
2/27	serum	Mumps	Compl. fix.	1:16
3/2	CSF	Trep. pallid.	Wassermann	negative
			Citochol	negative
			Kahn	negative
3/27	serum	Echo Virus types 4, 6, 9, 14	Neutralization	negative

Rectus femoris, Sartorius, Gastrocnemius, Thenar, and Interossei muscles were atrophic. An encephalomalacitic focus in the left Corpus striatum, with destruction of large areas of the Globus Pallidum, and Inner and Outer Capsules was found. The blood vessels at the base of the brain were arteriosclerotic. Other significant findings included bilateral confluent Bronchopneumonia, hemorrhagic infarction of the right lower lobe, thrombosis of the Femoral and Iliac veins, multiple embolie of the Pulmonary arteries, and arterio and arteriolosclerosis of the Kidneys. The Myocardium showed fragmentation, fatty infiltration, and brown atrophy, with dilatation of the right ventricle.

Conclusions

Comparison of the present case with those reviewed from the literature reveals the appearance of typical Ménièriform symptomatology in a somewhat older patient (66 years as opposed to 20–43). The Facial nerve was the first involved, as it was in five of the cases reviewed. Subsequently, contralateral Facial and bilateral Statoacoustic nerve lesions developed. In the cases seen above only three (11, 12, 15) showed bilateral dysfunction. Further spread of the process to the nerves of the extremities and trunk, as seen in our patient, was reported in only one of the cases reviewed (case 14). Unlike the fifteen cases noted above, there was no return of normal function in our patient. This could be attributed to a weakened and cachectic state in an older patient. The fatal outcome in our case was unrelated to the Polyneuritis, the patient developing cerebral thrombosis.

The most probable aetiological agent, in the case here presented, was Influenza, though antibody titers were normal. The patient reported an episode of Influenza preceeding the Neuritis. The serum determinations, however, were not made until two months after infection. Influenza antibody titers usually remain significantly elevated for only four weeks following initial clinical signs [15].

Examination of the cerebrospinal fluid revealed high protein values and moderate pleocytosis. If one were to apply the earlier criteria for the diagnosis of Guillain-Barré Syndrome to our case, it would not fit the definition of this disease. As noted above, Haymaker and Kernohan more broadly defined the clinical entity of 'Landry-Guillain-Barré Syndrome', allowing for variability in cerebrospinal fluid changes and prognosis. Our case is presented as an example of this syndrome, with somewhat atypical nerve involvement and course.

Summary

(1) A review of the original papers by Landry and Guillain, Barré, and Strohl, as well as subsequent analysis of this subject by other authors, reveal that the 'Landry' and 'Guillain-Barré' Syndromes are different manifestations of the same disease process.

(2) A description of 15 cases, seen in the literature, of a rare form of this process, simulating Ménières Disease, is presented.

(3) A case of 'Polyneuritis Cerebralis Ménièriformis', seen in the Neurological Institute, Basel, with bilateral involvement, and high spinal fluid protein and cells is described.

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